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#4352

Hck Antibody

For Research Use Only. Not for Use in Diagnostic Procedures.

Applications: W	Reactivity: H M Mk	Sensitivity: Endogenous	MW (kDa): 61	Source/Isotype: Rabbit	UniProt ID: #P08631	Entrez-Gene Id: 3055
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Product Usage Information

Application

Western Blotting

Dilution

1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Hck Antibody detects endogenous levels of total Hck protein. This antibody does not cross-react with family members Src, Lyn and Fyn.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to amino-terminal residues of human Hck. Antibodies are purified by protein A and peptide affinity chromatography

Background

The hematopoietic cell kinase (Hck) is a Src family protein tyrosine kinase that is prominently expressed in lymphoid and myeloid lineages of hematopoiesis (1). The Hck kinase participates in transduction of a variety of extracellular signals that affect cellular processes, including cell proliferation, differentiation, and migration. Hck protein structure includes a relatively divergent amino-terminal "unique" domain that is subject to post-translational lipid modifications and targets Hck to the plasma membrane. Src homology 3 (SH3) and 2 (SH2) domains, and a tyrosine kinase catalytic domain are adjacent to the "unique" domain. Research studies indicate that phosphorylation of conserved tyrosine residues positively and negatively regulate Hck catalytic activity. Phosphorylation of Hck at the conserved, carboxy-terminal Tyr499 by protein kinase Csk promotes an interaction between the phosphorylated tyrosine and the SH2 domain, rendering Hck inactive. Disruption of this interaction through dephosphorylation, the replacement of the Tyr522 with phenylalanine, or carboxy-terminal truncation mutations, results in constitutive activation of Hck. Autophosphorylation of Tyr411 within the kinase domain positively regulates Hck catalytic activity. Thus, the activation of Hck requires both disruption of the regulatory tyrosine-SH2 domain interaction and autophosphorylation of the regulatory tyrosine residue within the kinase domain (2,3). The dysfunction or dysregulation of Hck may contribute to the pathogenesis of some human forms of leukemia (4).

Background References

1. Karasek, M. et al. (1978) *Cell Tissue Res* 195, 547-56.
2. Ziegler, S.F. et al. (1989) *Mol Cell Biol* 9, 2724-7.
3. Kefalas, P. et al. (1995) *Int J Biochem Cell Biol* 27, 551-63.
4. Hu, Y. et al. (2004) *Nat Genet* 36, 453-61.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting

Cross-Reactivity Key

H: Human **M:** Mouse **Mk:** Monkey

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